Volume 4 | Issue 3 | 2024

ISSN | 2771-019X

Journal of
Clinical & Medical Images
Case Reports

Open Access | Case Report

Eosinophilic gastroenteritis: Rare disease, tricky diagnosis – Case presentation

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Abstract

We present a case of a 29 year old man who was admitted to our department due to abdominal pain and nausea. Furthermore, the patient presents fluid in the abdomen, thickening of duodenum wall and eosinophilia in peripheral blood. As a result of a complicated diagnostic process, he was diagnosed with eosinophilic gastroenteritis, which is associated with an ambiguous clinical presentation and diverse differential diagnostics.

Received: Mar 21, 2024

Accepted: Apr 29, 2024

Published Online: Apr 30, 2024

Cite this article: Szafranek S, Kanikowska A, Dobrowolska A, Eder P. Eosinophilic gastroenteritis: Rare disease, tricky diagnosis – Case presentation. J Clin Med Images Case Rep. 2024; 4(3): 1677.

Keywords: Eosinophilic gastroenteritis; Eosinophilia; Inflammatory; Abdominal fluid.

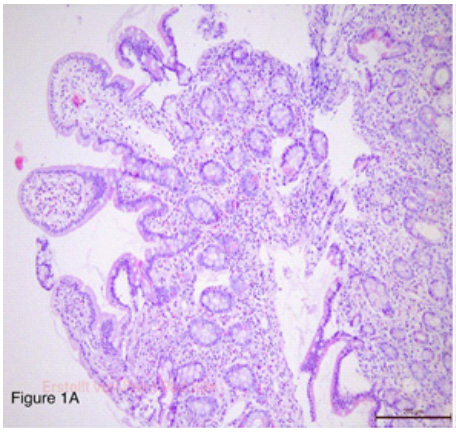
Introduction

Eosinophilic Gastroenteritis (EGE) is a rare inflammatory disease that leads to stomach and/or small intestine eosinophil infiltration and inflammatory symptoms [1]. While the cause remains unknown, many factors can trigger EGE. Klein classification divides EGE into three subtypes: mucosal, muscular and serosal and the symptoms depend on the inflamed part of the gastrointestinal tract wall [2,3]. Typical symptoms include abdominal pain, diarrhea, vomiting, nausea, intestinal obstruction and ascites. Exclusion of other causes of tissue eosinophilia and endoscopy with the biopsy is required to confirm the diagnosis. Histopathology EGE assessment is based on the examined section of gastrointestinal tract - for duodenum biopsy, it is >20 eosinophils/HPF (High Power Field). The treatment by choice are steroids, other treatments include a six-food elimination diet, leukotriene receptor antagonists, proton pump inhibitors, mast cell stabilizers, immunomodulators [4].

Case presentation

A 29-year- old man was admitted to the Department of Gastroenterology, Internal Diseases and Dietetics in 2021 due to severe abdominal pain and nausea for two preceding weeks, mild ascites and thickening of duodenum wall with enlarged lymph nodes detected in abdominal Computed Tomography (CT) (Figure 1B). The patient suffered from asthma treated with inhalant budesonide and formoterol.

On admission laboratory findings revealed blood eosinophilia 3,65x10^3/ul (reference range RR 0,02-0,5), percentage of eosinophils in blood smear 37% (RR 0,5-5,5%), elevated CRP concentration 13.2 mg/l (RR 5,0 mg/dl), elevated TSH level 6.4 µU/ml (WRR 0,27-4,2). The patient's general condition was good and the physical examination showed a painful upper abdominal area without rebound tenderness. The patient worked as a volunteer in an animal shelter for the past few months, therefore diagnostic blood and stool tests to exclude parasitic



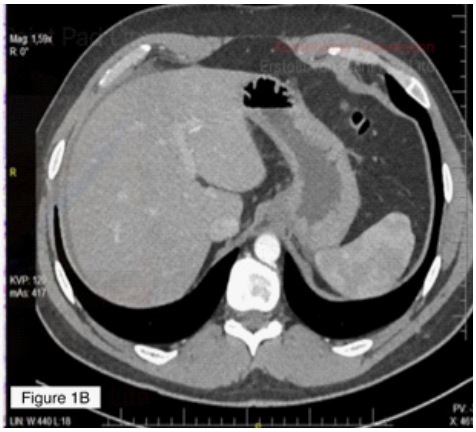


Figure 1: **A)** fragment of the proper plate of the duodenal mucous membrane with a chronic inflammatory process of low intensity focally present, with numerous eosinophils (over 55 per one large field of view, >55 eosinophils/1 LPF). **B)** Mild ascites and thickening of duodenum wall with enlarged lymph nodes detected in abdominal.

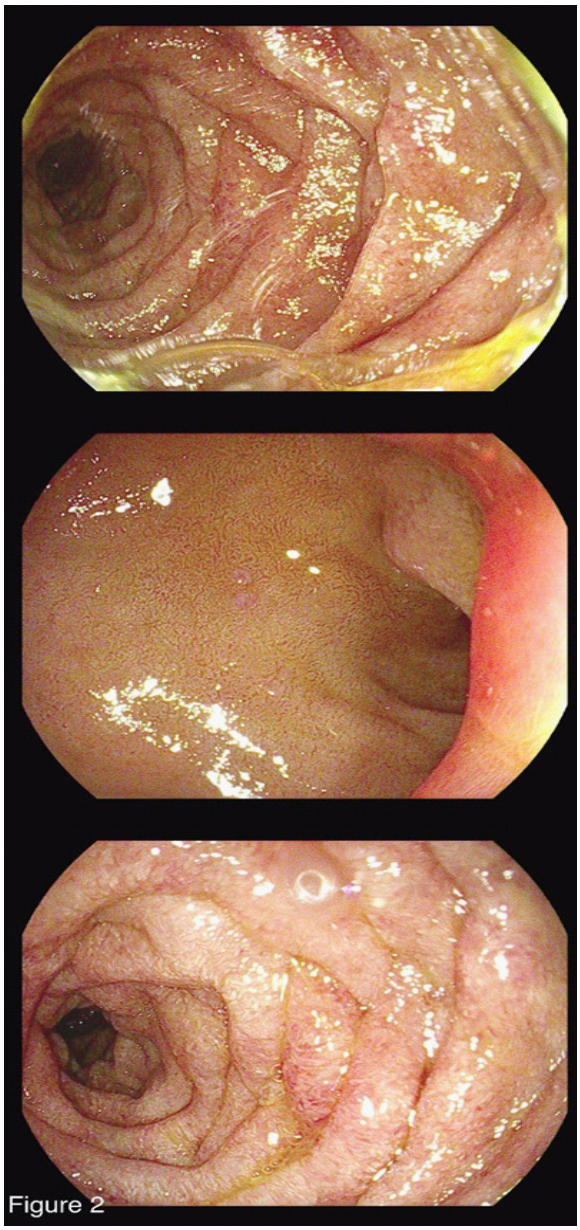


Figure 2: Linear furrows in the stomach and pyloric antrum with congested mucosa, enlarged and congested duodenal plicae circulares.

infestation were performed and did not show any positive results. Specific Ig-E dependent food allergy panel was negative.

Screening for the inflammatory and neoplastic causes of abdominal fluid was performed using the blood tests (HIV-1/HIV-2, hepatitis type B and C, cytomegalovirus, tuberculosis, Yersinia spp., p-ANCA, c-ANCA, ANA, anti-tTG), Ultrasound (US) and endoscopic procedures. The gastroscopy revealed: (Figure 2) linear furrows in the stomach and pyloric antrum with congested mucosa, enlarged and congested duodenal plicae circulares. Helicobacter pylori infection was detected. MR enterography showed submucosal edema of jejunum and ileum. Colonoscopy, echocardiography, thyroid and testicle US did not show any abnormalities. Eosinophilic gastroenteritis was confirmed by histopathological findings which revealed the inflammation with numerous eosinophils in the duodenum biopsy and focal fibrosis in the stomach biopsy (Figure 1A). At first H.pylori eradication-14-day standard therapy with bismuth subcitrate, tetracycline and metronidazole was implemented. Despite a successful H. pylori eradication, no reduction in the blood eosinophilia was observed and a small amount of fluid was still present in the peritoneal cavity in US. Oral prednisolone in 40 mg dose was administered for two weeks, with a gradual dose tapering for the next 6 weeks. The follow-up ultrasound examination reported no ascites in the abdomen and the blood eosinophils count was normal. In conclusion, the complex differential diagnosis led to right diagnosis and effective treatment, though the patient has to stay under regular follow-up to detect possible relapse.

Discussion/conclusion

The presented case highlights the diagnostic challenges encountered in identifying and managing Eosinophilic Gastroenteritis (EGE).

Lack of specific diagnostics markers and diverse clinical manifestation make the diagnostic process difficult. The presence of eosinophilia in peripheral blood and characteristic findings on imaging studies, such as submucosal edema detected on MR enterography, raised suspicion for EGE, although the final diagnosis can only be made based on histopathological results.

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